Modern Concepts of Cardiovascular Disease

Published monthly by the AMERICAN HEART ASSOCIATION

50 WEST 50TH STREET, NEW YORK, N. Y.

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Vol. IX **May, 1940** **No. 5**

EMBOLISM AND THROMBOSIS OF THE ABDOMINAL AORTA

Embolism and thrombosis of the abdominal aorta are unusual but very serious manifestations of cardiovascular disease. During the past fifty years approximately 138 cases of this condition have been reported in the medical literature. Of the 138 patients reported, only 23 survived. Of these 23, 9 were relieved by embolectomy and the remaining 14 recovered under conservative medical management. In the majority of cases death was not long delayed, following close upon gangrene of one or both legs. Hesse in a study of 46 cases found that 52% had died within one week and all were dead within six months. The author was able to follow three cases that survived one, three, and five years respectively after the onset of initial embolic symptoms. Two of the three patients died of intercurrent disease without developing gangrene.

Embolism and thrombosis of the abdominal aorta occur most frequently during the course of chronic cardiovalvular disease, the great majority of cases being associated with mitral stenosis and auricular fibrillation. It is a condition usually seen in the middle decades of life though cases have been reported at all ages. The youngest case reported was that of a ten-day old infant with an umbilical cord infection. A few similar cases have been reported as occurring during or following an acute infection.

The terms embolism and thrombosis have been used together in the literature advisedly, inasmuch as the two conditions are intimately associated from the pathological aspects. It is difficult and sometimes impossible to determine whether the obstructing clot in the aorta is embolus, thrombus, or both. On microscopic examination, the endothelium of the vessel is found to be destroyed, the media is engorged, there is a reduplication of the small vessels and an excess of phagocytes containing hemosiderin.

The symptomatology of these cases varies according to the size of the embolus, the site at which it lodges, the degree of secondary thrombosis which ensues, and finally the adequacy of the collateral circulation that develops. The most dramatic type of this condition is that due to a large embolus arising from a cardiac chamber and lodging at the bifurcation of the aorta into the common iliac arteries. The embolus then grows by the process of extension, termed "secondary thrombosis," to involve the lumen of the vessel immediately above and, to a less extent, below the original obstruction. The clinical manifestation of this type of involvement is the sudden onset of excruciating pain in the mid-abdomen or back, followed in a few minutes by a severe ache in one or both legs. The ache is shortly followed by numbness and complete loss of sensation as death of tissue occurs. The patient presents the appearance of extreme shock. The lower extremities are cold and pallid. Arterial pulsations are lost throughout the leg. Death usually occurs within a few days, shortly after the development of gangrene of one or both legs. This type of case is usually not difficult to diagnose and is the variety most often seen.

When the primary embolus is smaller, the symtomatology is somewhat different. The small (and frequently multiple) emboli lodge in the arteries of the lower portion of the leg, the posterior tibial artery being the most frequent site. Due to the obstruction in this artery a column of stationery blood develops above, and a retrograde thrombus forms at a variable distance and at a variable speed above the site of the primary obstruction. The thrombus extends up through the femoral and iliac arteries to the bifurcation of the aorta where a piece may be dislodged into the iliac artery of the other side. The thrombus may continue up the aorta to involve the arteries branching from this vessel. The symptoms in this type of case depend upon the balance between impairment of the lumen of the vessels and the development of a collateral circulation. If the collateral circulation is poor, gangrene of the foot may develop, associated with moderate shock and toxemia. The collateral circulation in a few cases may become so well established that a complete obstruction of the abdominal aorta may be present and the patient complain only of intermittent claudication. Two such cases have been seen by the author. One was a woman of middle age who

suffered from rheumatic heart disease with mitral stenosis and auricular fibrillation. Death was due to a cerebral embolus. Twelve months prior to death an embolus had lodged in the left leg. Recovery was so complete that the patient was able to be up and around and had complained of no symptoms except the presence of intermittent claudication in both legs. At post mortem examination, the abdominal aorta and both iliac arteries were thrombosed throughout their entire diameter. The clot had extended in the aorta to the level of the renal arteries. By microscopic examination it was shown to be old and well organized.

A similar case was observed in a man of 21 years who, at autopsy, was found to have a congenitally bicuspid aortic valve with small organized vegetations on one of the cusps. In this case, the aorta was thrombosed to the level of the diaphragm, with involvement of the celiac artery, one renal artery, and both superior and inferior mesenteric arteries. The thrombus extended downward into both iliac arteries. Sections taken through the thrombus failed to show any evidence of recanalization. The history of this case was that three years before the patient had noted tightening of the muscles of the legs. This symptom was followed two weeks later by severe upper abdominal pain associated with nausea and vomiting. The abdominal pain subsided within three days and in a few more days, he was able to be up. Since that time he had complained of persistent intermittent claudication of both legs as the only pertinent symptom. Death in this case was due to multiple pulmonary infarcts originating from a thrombosis of the inferior vena cava. In both the above mentioned cases, a fair collateral circulation was demonstrable at autopsy.

The third and least common type is obstruction of the abdominal aorta by a primary thrombus arising on an arteriosclerotic basis. The thrombus may develop in the abdominal aorta itself, or it may originate in a smaller distal vessel and extend to the abdominal aorta by a retrograde process. This type of case, like that due to small emboli, presents a pattern of symptoms that is often unrecognized. Intermittent claudication is usually the first complaint, followed by numbness, coldness, pallor, cyanosis, and finally gangrene of the legs. However, as all these symptoms indicate only impaired blood supply to the legs, they are of no value in demonstrating the level of the obstruction or in localizing the lesion. Palpation of the femoral arteries will demonstrate whether or not the obstruction has involved these vessels. When the abdominal wall is thin, the absence of pulsation of the abdominal aorta may be appreciated by direct palpation, and occasionally a swelling may be felt at the upper limit of the thrombus. It must be kept in mind that immediately below the umbilicus the aorta divides and dips into the pelvis so that normally no pulsation can be felt below this level.

The course of the disease and the symptoms depend on three factors. First, the size and position of the original insult to the circulation; generally, the larger the vessel originally occluded the greater the probability of development of early gangrene. Second, the ability of the collateral circulation to keep pace with diminishing circulation through the usual channels; — the collateral circulation depends upon the elasticity of adjoining blood vessels and a fairly slow obstructing mechanism. Third, and equally important, is the primary disease process from which the emboli or thrombi arise; for example, the presence of fibrillation of the auricles or a mural thrombus of the ventricular wall following coronary thrombosis. Rykert and Graham believe that the primary disease, and not the immediate restoration of peripheral circulation, is the most important factor in determining the late prognosis.

The treatment of this type of disease is divided into surgical and medical, and considerable difference exists as to their respective merits. Surgical interference in the form of embolectomy is indicated in the selected case in which the process is known to be of sudden onset and the embolus is large. The operation must be performed within the first twelve hours if success is to be obtained. After that length of time, so much damage has occurred to the surrounding arterial wall that a thrombus will reform at the operative site. Even with the most fortunate selection of cases the operation, either upon the femoral artery or the abdominal aorta, is successful in only about 20% of the cases. It is also difficult to determine within the first twelve hours how efficiently a collateral circulation may be established later if embolectomy is not performed. Tingling in the toes after the appearance of numbness is a symptom of returning blood supply and warrants the continuation of expectant treatment. This consists of the usual therapy for shock combined with the early application of alternating suction and pressure to the affected limb. If the early signs of improvement in circulation develop, partially occlusive bandages, Buerger's exercises, the oscillating bed, tissue extracts, alcohol and papaverine may be used to advantage to promote the production of collateral circulation.

In summary, a type of occlusive disease of the aorta has been discussed which produces a very high mortality. This exhibits a varied symptom pattern ranging from sudden onset of severe shock followed shortly by bilateral gangrene of the legs, to an onset with intermittent claudication as the first and, occasionally, the only symptom.

The wide range of symptoms depends upon the size of the primary embolus, the site of its lodgement, and the rapidity of the development of a retrograde thrombus above it. The severity of the underlying disease process is of the greatest importance in determining the late prognosis.

Embolectomy should be considered when the diagnosis of a large embolus can be definitely established within a few hours after the onset of symptoms in a patient in whom a similar vascular accident is not to be expected again within a short time. On the other hand, the results of conservative medical treatment appear to be as good as, if not better than, those obtained from the more radical surgical procedures.

When the occluding process is gradual, the course of the disease is insidious and presents a difficult diagnostic problem. Intermittent claudication is one constant symptom, and the development of gangrene may be long delayed or absent in spite of complete obliteration of the usual arterial channels. This type of case illustrates the ability of the circulatory system to adapt itself to a gradually occluding process and emphasizes the fact that extensive pathological changes may occur in the large vessels of the body with only minor symptoms.

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